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NEWS 7	NOV 03	New format for Korean patent application numbers in CA/CAplus increases consistency, saves time.
NEWS 8	NOV 04	Selected STN databases scheduled for removal on December 31, 2010
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NEWS 10	NOV 22	Higher System Limits Increase the Power of STN Substance-Based Searching
NEWS 11	NOV 24	Search an additional 46,850 records with MEDLINE backfile extension to 1946
NEWS 12	DEC 14	New PNK Field Allows More Precise Crossover among STN Patent Databases
NEWS 13	DEC 18	ReaxysFile available on STN
NEWS 14	DEC 21	CAS Learning Solutions -- a new online training experience
NEWS 15	DEC 22	Value-Added Indexing Improves Access to World Traditional Medicine Patents in CAplus
NEWS 16	JAN 24	The new and enhanced DPCI file on STN has been released
NEWS 17	JAN 26	Improved Timeliness of CAS Indexing Adds Value to USPATFULL and USPAT2 Chemistry Patents
NEWS 18	JAN 26	Updated MeSH vocabulary, new structured abstracts, and other enhancements improve searching in STN reload of MEDLINE
NEWS 19	JAN 28	CABA will be updated weekly
NEWS 20	FEB 23	PCTFULL file on STN completely reloaded
NEWS 21	FEB 23	STN AnaVist Test Projects Now Available for Qualified Customers
NEWS 22	FEB 25	LPCI will be replaced by LDPCI
NEWS 23	MAR 07	Pricing for SELECTing Patent, Application, and Priority Numbers in the USPAT and IFI Database Families is Now Consistent with Similar Patent Databases on STN
NEWS 24	APR 26	Expanded Swedish Patent Application Coverage in CA/CAplus Provides More Current and Complete Information
NEWS 25	APR 28	The DWPI (files WPINDEX, WPIDS and WPIX) on STN have been enhanced with thesauri for the European Patent Classifications

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 NEWS 26  MAY 02  MEDLINE Improvements Provide Fast and Simple Access to DOI and
                  Chemical Name Information
 NEWS 27  MAY 12  European Patent Classification thesauri added to the INPADOC
                  files, PCTFULL, GBFULL and FRFULL
 NEWS 28  MAY 20  PATDPA database updates to end in June 2011

 NEWS EXPRESS 17 DECEMBER 2010 CURRENT WINDOWS VERSION IS V8.4.2 .1,
          AND CURRENT DISCOVER FILE IS DATED 24 JANUARY 2011.

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FILE 'HOME' ENTERED AT 11:52:55 ON 20 MAY 2011

=> file medline
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                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.23       0.23

FILE 'MEDLINE' ENTERED AT 11:53:08 ON 20 MAY 2011

 FILE LAST UPDATED: 19 May 2011 (20110519/UP).  FILE COVERS 1946 TO DATE.

 MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject
 Headings (MeSH) vocabulary and tree numbers from the U.S.  National Library
 of Medicine (NLM).  Additional information is available at:

 http://www.nlm.nih.gov/pubs/techbull/nd10/nd10_medline_data_changes_2011.html.

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 substance identification.

 See HELP RANGE before carrying out any RANGE search.

=> s catenin and (protein kinase c or pkc)
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          12785 CATENIN
           1208 CATENINS
          12977 CATENIN
                (CATENIN OR CATENINS)
        2210493 PROTEIN
        1782640 PROTEINS
        2740349 PROTEIN
                (PROTEIN OR PROTEINS)
         337855 KINASE
         199825 KINASES
         384940 KINASE
                (KINASE OR KINASES)
        1389122 C
          52343 PROTEIN KINASE C
                (PROTEIN(W)KINASE(W)C)
          26271 PKC
           1623 PKCS
          27117 PKC
                (PKC OR PKCS)
L1          227 CATENIN AND (PROTEIN KINASE C OR PKC)

=> s l1 and iota or lambda
           1098 IOTA
              1 IOTAS
           1099 IOTA
                (IOTA OR IOTAS)
          39130 LAMBDA
             91 LAMBDAS
          39179 LAMBDA
                (LAMBDA OR LAMBDAS)
L2        39179 L1 AND IOTA OR LAMBDA

=> s l1 and (iota or lambda)
           1098 IOTA
              1 IOTAS
           1099 IOTA
                (IOTA OR IOTAS)
          39130 LAMBDA
             91 LAMBDAS
          39179 LAMBDA
                (LAMBDA OR LAMBDAS)
L3            6 L1 AND (IOTA OR LAMBDA)

=> d bib ab 1-6

L3   ANSWER 1 OF 6     MEDLINE on STN
AN   2009319691     MEDLINE
DN   PubMed ID: 19290490
TI   Expression of P-aPKC-iota, E-cadherin, and beta-catenin related to
     invasion and metastasis in hepatocellular carcinoma.
AU   Du Guang-Sheng; Wang Jian-Ming; Lu Jin-Xi; Li Qiang; Ma Chao-Qun; Du
     Ji-Tao; Zou Sheng-Quan
CS   Department of General Surgery, Tongji Hospital, Tongji Medical College,
     Huazhong University of Science and Technology, Wuhan, Hubei, China.
SO   Annals of surgical oncology, (2009 Jun) Vol. 16, No. 6, pp. 1578-86.
     Electronic Publication: 2009-03-17.
     Journal code: 9420840. E-ISSN: 1534-4681. L-ISSN: 1068-9265.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
     (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA   English
FS   Priority Journals
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EM 200906
ED Entered STN: 6 May 2009
Last Updated on STN: 19 Jun 2009
Entered Medline: 18 Jun 2009
OSC.G 1 There are 1 MEDLINE records that cite this record
AB OBJECTIVES: Atypical protein kinase C iota (aPKC-iota) and its associated intracellular molecules, E-cadherin and beta-catenin, are important for cell polarization in tumorigenesis and progression. Expression of aPKC-iota, P-aPKC-iota (activated aPKC-iota), E-cadherin, and beta-catenin in hepatocellular carcinoma (HCC) was measured, and correlation with clinicopathological characteristics of HCC was analyzed.

METHODS: Paraffin-embedded tumor tissue was obtained from patients with HCC after resection without preoperative radiotherapy or chemotherapy. Gene expression was detected by polymerase chain reaction (PCR), and protein expression was detected by immunohistochemistry and Western blot analysis. Expressions of aPKC-iota, P-aPKC-iota, E-cadherin, and beta-catenin were analyzed with relation to the clinicopathological data.

RESULTS: The gene and protein expression of aPKC-iota are obviously higher in HCC tissues than that in peritumoral tissues and normal tissues by semiquantitative PCR and immunohistochemistry methods. Accumulation of aPKC-iota in HCC cytoplasm and nucleolus inhibited the later formation of belt-like adherens junctions (AJs) and/or tight junctions (TJs) in cell-cell contact. E-cadherin was reduced and accumulation of cytoplasm beta-catenin was increased in HCC. The expression of aPKC-iota was closely related to pathological differentiation, tumor size, invasion, and metastasis of HCC.

CONCLUSION: Accumulation of cytoplasm aPKC-iota may reflect pathological differentiation, invasion, and metastasis potential of HCC. In this regard, our study on HCC revealed the potential usefulness of aPKC-iota, E-cadherin, and beta-catenin as a prognostic marker, closely related to pathological differentiation, invasion, metastasis, and prognosis of HCC.

L3 ANSWER 2 OF 6 MEDLINE on STN
AN 2009075089 MEDLINE
DN PubMed ID: 19147581
TI Protein kinase C betaII and PKCiota/lambda: collaborating partners in colon cancer promotion and progression.
AU Murray Nicole R; Weems Justin; Braun Ursula; Leitges Michael; Fields Alan P
CS Department of Cancer Biology, Mayo Clinic College of Medicine, Jacksonville, Florida 32224, USA.
NC CA081436 (United States NCI NIH HHS)
CA094122 (United States NCI NIH HHS)
R01 CA081436-11 (United States NCI NIH HHS)
SO Cancer research, (2009 Jan 15) Vol. 69, No. 2, pp. 656-62.
Journal code: 2984705R. E-ISSN: 1538-7445. L-ISSN: 0008-5472.
Report No.: NLM-NIHMS79085; NLM-PMC2688739.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
LA English
FS Priority Journals
EM 200902
ED Entered STN: 17 Jan 2009
Last Updated on STN: 24 Feb 2009
Entered Medline: 20 Feb 2009
OSC.G 3 There are 3 MEDLINE records that cite this record

REM.CNT 23 There are 23 cited references available in MEDLINE for this document.

AB We previously showed that elevated expression of either protein kinase CbetaII (PKCbetaII) or PKCiota/lambda enhances colon carcinogenesis in mice. Here, we use novel bitransgenic mice to determine the relative importance of PKCbetaII and PKCiota/lambda in colon carcinogenesis in two complimentary models of colon cancer in vivo. Bitransgenic mice overexpressing PKCbetaII and constitutively active PKCiota (PKCbetaII/caPKCiota) or kinase-deficient, dominant-negative PKCiota (PKCbetaII/kdPKCiota) in the colon exhibit a similar increase in colon tumor incidence, tumor size, and tumor burden in response to azoxymethane (AOM) when compared with nontransgenic littermates. However, PKCbetaII/kdPKCiota mice develop predominantly benign colonic adenomas, whereas PKCbetaII/caPKCiota mice develop malignant carcinomas. In contrast, PKCbeta-deficient (PKCbeta(-/-)) mice fail to develop tumors even in the presence of caPKCiota. Our previous data indicated that PKCbetaII drives tumorigenesis and proliferation by activating beta-catenin/Apc signaling. Consistent with this conclusion, genetic deletion of PKCbeta has no effect on spontaneous tumorigenesis in Apc(min/+) mice. In contrast, tissue-specific knockout of PKClambda significantly suppresses intestinal tumor formation in Apc(min/+) mice. Our data show that PKCbetaII and PKCiota/lambda serve distinct, nonoverlapping functions in colon carcinogenesis. PKCbetaII is required for AOM-induced tumorigenesis but is dispensable for tumor formation in Apc(Min/+) mice. PKCiota/lambda promotes tumor progression in both AOM- and Apc(min/+)-induced tumorigenesis. Thus, PKCbetaII and PKCiota, whose expression is elevated in both rodent and human colon tumors, collaborate to drive colon tumor formation and progression, respectively.

L3 ANSWER 3 OF 6 MEDLINE on STN
AN 2008503980 MEDLINE
DN PubMed ID: 18466329
TI Regulation of neural progenitor cell motility by ceramide and potential implications for mouse brain development.
AU Wang Guanghu; Krishnamurthy Kannan; Chiang Ying-Wei; Dasgupta Somsankar; Bieberich Erhard
CS Program in Developmental Neurobiology, Institute of Molecular Medicine and Genetics, School of Medicine, Medical College of Georgia, Augusta, Georgia, USA.
NC R01NS046835 (United States NINDS NIH HHS)
SO Journal of neurochemistry, (2008 Jul) Vol. 106, No. 2, pp. 718-33. Electronic Publication: 2008-05-02.
Journal code: 2985190R. E-ISSN: 1471-4159. L-ISSN: 0022-3042.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
LA English
FS Priority Journals
EM 200809
ED Entered STN: 9 Aug 2008
Last Updated on STN: 17 Sep 2008
Entered Medline: 16 Sep 2008
OSC.G 1 There are 1 MEDLINE records that cite this record

AB We provide evidence that the sphingolipid ceramide, in addition to its pro-apoptotic function, regulates neural progenitor (NP) motility in vitro and brain development in vivo. Ceramide (N-palmitoyl d-erythro sphingosine and N-oleoyl d-erythro sphingosine) and the ceramide analog N-oleoyl serinol (S18) stimulate migration of NPs in scratch (wounding) migration assays. Sphingolipid depletion by inhibition of de novo ceramide biosynthesis, or ceramide inactivation using an anti-ceramide antibody, obliterates NP motility, which is restored by ceramide or S18. These results suggest that ceramide is crucial for NP motility. Wounding

of the NP monolayer activates neutral sphingomyelinase indicating that ceramide is generated from sphingomyelin. In membrane processes, ceramide is co-distributed with its binding partner atypical protein kinase C zeta/lambda (aPKC), and Cdc42, alpha/beta-tubulin, and beta-catenin, three proteins involved in aPKC-dependent regulation of cell polarity and motility. Sphingolipid depletion by myriocin prevents membrane translocation of aPKC and Cdc42, which is restored by ceramide or S18. These results suggest that ceramide-mediated membrane association of aPKC/Cdc42 is important for NP motility. In vivo, sphingolipid depletion leads to ectopic localization of mitotic or post-mitotic neural cells in the embryonic brain, while S18 restores the normal brain organization. In summary, our study provides novel evidence that ceramide is critical for NP motility and polarity in vitro and in vivo.

L3 ANSWER 4 OF 6 MEDLINE on STN
AN 2006668260 MEDLINE
DN PubMed ID: 16959241
TI Beta-catenin is essential for lamination but not neurogenesis in mouse retinal development.
AU Fu Xueyao; Sun Hongxia; Klein William H; Mu Xiuqian
CS Department of Biochemistry and Molecular Biology, Unit 1000, The University of Texas M. D. Anderson Cancer Center, 1515 Holcombe Blvd., Houston, TX 77030, USA.
NC CA16672 (United States NCI NIH HHS)
EY011930 (United States NEI NIH HHS)
R01 EY011930-07 (United States NEI NIH HHS)
R01 EY011930-08 (United States NEI NIH HHS)
R01 EY011930-09 (United States NEI NIH HHS)
SO Developmental biology, (2006 Nov 15) Vol. 299, No. 2, pp. 424-37.
Electronic Publication: 2006-08-10.
Journal code: 0372762. ISSN: 0012-1606. L-ISSN: 0012-1606.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200701
ED Entered STN: 21 Nov 2006
Last Updated on STN: 17 Jan 2007
Entered Medline: 16 Jan 2007
OSC.G 12 There are 12 MEDLINE records that cite this record
AB During vertebrate retinal development, the seven retinal cell types differentiate sequentially from a single population of retinal progenitor cells (RPCs) and organize themselves into a distinct laminar structure. The purpose of this study was to determine whether beta-catenin, which functions both as a nuclear effector for the canonical Wnt signaling pathway and as a regulator of cell adhesion, is required for retinal neurogenesis or lamination. We used the Cre-loxP system to either eliminate beta-catenin or to express a constitutively active form during retinal neurogenesis. Eliminating beta-catenin did not affect cell differentiation, but did result in the loss of the radial arrangement of RPCs and caused abnormal migration of differentiated neurons. As a result, the laminar structure was massively disrupted in beta-catenin-null retinas, although all retinal cell types still formed. In contrast to other neural tissues, eliminating beta-catenin did not significantly reduce the proliferation rate of RPCs; likewise, activating beta-catenin ectopically in RPCs did not result in overproliferation, but loss of neural retinal identity. These results indicate that beta-catenin is essential during retinal neurogenesis as a regulator of cell adhesion but not as a nuclear effector of the canonical Wnt signaling pathway. The results further imply that retinal lamination and retinal

cell differentiation are genetically separable processes.

L3 ANSWER 5 OF 6 MEDLINE on STN
AN 2006345827 MEDLINE
DN PubMed ID: 16478782
TI Regulation of dishevelled and beta-catenin in rat skeletal muscle: an alternative exercise-induced GSK-3beta signaling pathway.
AU Aschenbach William G; Ho Richard C; Sakamoto Kei; Fujii Nobuharu; Li Yangfeng; Kim Young-Bum; Hirshman Michael F; Goodyear Laurie J
CS Joslin Diabetes Center, One Joslin Place, Boston, MA 02215, USA.
NC F32-AR-049662 (United States NIAMS NIH HHS)
F32-DK-59769 (United States NIDDK NIH HHS)
R01-AR-42238 (United States NIAMS NIH HHS)
R01-DK-68626 (United States NIDDK NIH HHS)
SO American journal of physiology. Endocrinology and metabolism, (2006 Jul) Vol. 291, No. 1, pp. E152-8. Electronic Publication: 2006-02-14. Journal code: 100901226. ISSN: 0193-1849. L-ISSN: 0193-1849.
CY United States
DT (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
LA English
FS Priority Journals
EM 200608
ED Entered STN: 9 Jun 2006
Last Updated on STN: 4 Aug 2006
Entered Medline: 3 Aug 2006
OSC.G 2 There are 2 MEDLINE records that cite this record
AB beta-catenin is a multifunctional protein involved in cell-cell adhesion and the Wnt signaling pathway. beta-Catenin is activated upon its dephosphorylation, an event triggered by Dishevelled (Dvl)-mediated phosphorylation and deactivation of glycogen synthase kinase-3beta (GSK-3beta). In skeletal muscle, both insulin and exercise decrease GSK-3beta activity, and we tested the hypothesis that these two stimuli regulate beta-catenin. Immunoblotting demonstrated that Dvl, Axin, GSK-3beta, and beta-catenin proteins are expressed in rat red and white gastrocnemius muscles. Treadmill running exercise in vivo significantly decreased beta-catenin phosphorylation in both muscle types, with complete dephosphorylation being elicited by maximal exercise. beta-Catenin dephosphorylation was intensity dependent, as dephosphorylation was highly correlated with muscle glycogen depletion during exercise ($r(2) = 0.84$, $P < 0.001$). beta-Catenin dephosphorylation was accompanied by increases in GSK-3beta Ser(9) phosphorylation and Dvl-GSK-3beta association. In contrast to exercise, maximal insulin treatment (1 U/kg body wt) had no effect on skeletal muscle beta-catenin phosphorylation or Dvl-GSK-3beta interaction. In conclusion, exercise in vivo, but not insulin, increases the association between Dvl and GSK-3beta in skeletal muscle, an event paralleled by beta-catenin dephosphorylation.

L3 ANSWER 6 OF 6 MEDLINE on STN
AN 2005255330 MEDLINE
DN PubMed ID: 15665047
TI Potential candidates for ischemic preconditioning-associated vascular growth pathways revealed by antibody array.
AU Mathur Praveer; Kaga Shigeaki; Zhan Lijun; Das Dipak K; Maulik Nilanjana
CS Molecular Cardiology Laboratory, Dept. of Surgery, Univ. of Connecticut School of Medicine, Farmington, CT 06030-1110, USA.
NC HL-22559 (United States NHLBI NIH HHS)
HL-33889 (United States NHLBI NIH HHS)
HL-56803 (United States NHLBI NIH HHS)
HL-69910 (United States NHLBI NIH HHS)
SO American journal of physiology. Heart and circulatory physiology, (2005

Jun) Vol. 288, No. 6, pp. H3006-10. Electronic Publication: 2005-01-21.
Journal code: 100901228. ISSN: 0363-6135. L-ISSN: 0363-6135.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LA English
FS Priority Journals
EM 200507
ED Entered STN: 18 May 2005
Last Updated on STN: 8 Jul 2005
Entered Medline: 7 Jul 2005
OSC.G 3 There are 3 MEDLINE records that cite this record
AB Our understanding of the phenomenon of myocardial vascular growth is very limited even though various studies have been conducted in several different models, because the focus in each has been on a select very few number of proteins as the possible growth factors. In the present study, we used the ischemic preconditioning (IP) model in the form of four in vivo repetitive cycles of coronary artery occlusion, each followed by reperfusion as the model to stimulate vascular growth, and performed the protein profiling using high-throughput antibody array technology. Rats were divided into two groups: control + left anterior descending coronary artery (LAD) occlusion (CMI), and IP+ LAD occlusion (IPMI). The antibody array experiment performed to compare the expression of 512 proteins between the IPMI and CMI samples revealed significant upregulation of growth proteins like TGF-beta, BMX, granulocyte-monocyte colony-stimulating factor, signal transducer and activator of transcription 3, alpha- and beta-catenins, ubiquitin-conjugating enzyme UbcH6, nexilin, and PKC-epsilon and -lambda. JNK1 and c-Src tyrosine kinase were expectedly found to be downregulated. Western blot experiments validated the changes in expression of these proteins. Therefore, this study puts forward the above-mentioned proteins as valid participants in the vascular growth signals that are known to be triggered by ischemic preconditioning of heart.

=> d his full

(FILE 'HOME' ENTERED AT 11:52:55 ON 20 MAY 2011)

FILE 'MEDLINE' ENTERED AT 11:53:08 ON 20 MAY 2011
L1 227 SEA PLU=ON CATENIN AND (PROTEIN KINASE C OR PKC)
L2 39179 SEA PLU=ON L1 AND IOTA OR LAMBDA
L3 6 SEA PLU=ON L1 AND (IOTA OR LAMBDA)
D BIB AB 1-6

FILE HOME

FILE MEDLINE
FILE LAST UPDATED: 19 May 2011 (20110519/UP). FILE COVERS 1946 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Libra of Medicine (NLM). Additional information is available at:

http://www.nlm.nih.gov/pubs/techbull/nd10/nd10_medline_data_changes_2011.

The 2011 Medline reload was completed on January 22, 2011.
See HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate

```
      substance identification.

      See HELP RANGE before carrying out any RANGE search.

=> s catenin and apkc and iota
         12785 CATENIN
          1208 CATENINS
         12977 CATENIN
                 (CATENIN OR CATENINS)
           414 APKC
            60 APKCS
           431 APKC
                 (APKC OR APKCS)
          1098 IOTA
             1 IOTAS
          1099 IOTA
                 (IOTA OR IOTAS)
L4           1 CATENIN AND APKC AND IOTA

=> s l4 not l3
L5           0 L4 NOT L3

=> d his full

     (FILE 'HOME' ENTERED AT 11:52:55 ON 20 MAY 2011)

     FILE 'MEDLINE' ENTERED AT 11:53:08 ON 20 MAY 2011
L1         227 SEA PLU=ON  CATENIN AND (PROTEIN KINASE C OR PKC)
L2       39179 SEA PLU=ON  L1 AND IOTA OR LAMBDA
L3           6 SEA PLU=ON  L1 AND (IOTA OR LAMBDA)
               D BIB AB 1-6
L4           1 SEA PLU=ON  CATENIN AND APKC AND IOTA
L5           0 SEA PLU=ON  L4 NOT L3


     FILE HOME

     FILE MEDLINE
      FILE LAST UPDATED: 19 May 2011 (20110519/UP).  FILE COVERS 1946 TO DATE.

      MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject
      Headings (MeSH) vocabulary and tree numbers from the U.S.  National Libra
      of Medicine (NLM).  Additional information is available at:

      http://www.nlm.nih.gov/pubs/techbull/nd10/nd10_medline_data_changes_2011.

      The 2011 Medline reload was completed on January 22, 2011.
      See HELP RLOAD for details.

      This file contains CAS Registry Numbers for easy and accurate
      substance identification.

      See HELP RANGE before carrying out any RANGE search.

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COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                 ENTRY    SESSION
FULL ESTIMATED COST                               5.34       5.57

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